Whole Heart Flow Sensitive 4D MRI in Congenital Heart Disease

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Introduction: Flow sensitive 4D MR imaging has been shown to be a useful tool for the assessment of vascular hemodynamics [1]. Previous studies focused on flow characterization of the aorta in healthy volunteers and comparison of normal flow patterns with hemodynamic alterations in aortic pathologies [2,3]. Initial results of complex vascular hemodynamics in congenital heart disease have recently been reported [4]. The purpose of this study was to employ whole heart 4D MRI for the analysis of vessel morphology and altered hemodynamics in patients with operated congenital heart disease, particularly with respect to changes on the pulmonary system.

Methods: Measurements were performed in 15 patients (age range: 2-26 years, 6 female, 9 male) on a 1.5T MR system (n=11, Avanto, Siemens, Germany) and on a 3T MRI (n=4, TRIO, Siemens, Germany). The study included 5 patients after repair of tetralogy of Fallot, 2 patients after transposition of the great arteries (D-TGA) and 8 patients after Glenn-operation or extracardiac total cavopulmonary connection (TCPC). Data acquisition included the entire heart as well as the adjacent large arterial and venous vessels and was performed during free breathing using prospective ECG gating and respiration control. The MRI sequence consisted of a k-space segmented rf-spoiled gradient echo sequence with interleaved 3-directional velocity encoding (venc = 150-200cm/s, TE/TR = 2.4ms/4.8ms, $\alpha = 7°$, FOV = 320x240mm, spatial resolution = 2.5x2.5x2.8mm³, temporal resolution = 38.4ms, scan time ~ 10-20min). Data analysis included the calculation of a 3D phase contrast (PC) MR angiogram (MRA) from the 4D MR data which was combined with 3D blood flow visualization (EnSight v. 8.2, CEI, Apex, NC, USA) [5]. The description of postoperative altered hemodynamics was achieved by traces of 3D virtual particles demonstrating time-resolved blood flow. Particle traces were emitted from interactively positioned planes from which traces along the measured velocities could be color coded according to the local velocity or by their anatomic origin (3D flow connectivity mapping) to identify the blood flow paths with respect to their vascular origin.

Results: The postoperative changes in hemodynamics could be well visualized in all cases. The main MR-findings of the individually observed blood flow abnormalities are summarized in table 1. In patients after transposition of the great arteries, obvious flow acceleration was observed in the pulmonary arteries without any stenosis or dilatation. No abnormality was detected in the aorta (Fig. 1). The method clearly visualized post-operative blood flow originating in the superior and inferior venae cavae and its distribution to the right and left pulmonary artery in patients with TCPC using connectivity mapping (Fig.2). Disturbed flow dynamics during the phase of ventricular filling due to moderate to severe retrograde flow caused by pulmonary insufficiency was observed in patients after repair of tetralogy of Fallot. Substantial systolic vortices were detected in the ectatic outflow tract and in the main pulmonary artery as well as in the left pulmonary artery (Fig. 3). Distinctive complex blood flow patterns and vortex formation in the right atrium and ventricle could be visualized in patients with hypoplastic left heart syndrome in the state of Norwood 2 (not shown).

Discussion: Preliminary results from this study indicate that whole heart 4D flow sensitive MRI and time-resolved 3D velocity mapping permits the assessment of blood flow characteristics in patients after repair of congenital heart disease even at 1.5T. Although further larger studies including additional quantitative data will be needed for the final evaluation of diagnostic impact of this technique, these results illustrate the feasibility of in-vivo visualization of dynamic blood flow. The complete spatial and temporal coverage of the entire heart and surrounding vessels provides a better understanding of the extraordinary cardiac and vessel morphology as well as the altered flow patterns in these patients and facilitates an early recognition of pathologies which need intervention.

References: 1. Wigstrom L, et al. Magn Reson Med 1999;41(4):793-799 **2.** Markl M, et al. J Comput Assist Tomogr 2004;28(4):459-468. **3.** Frydrychowicz A, et al. J Thorac Cardiovasc Surg 2008;136(2):400-407 **4.** Frydrychowicz A, et al. Circulation 2008; e118 (2):16-17. **5.** Markl M, et al. JMRI 2007;25:824-831.

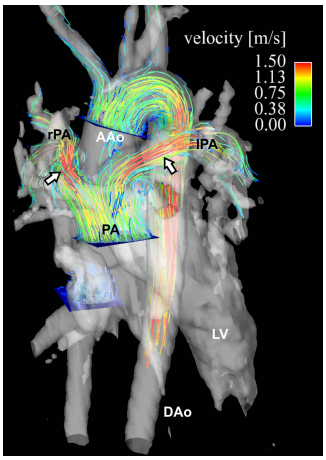
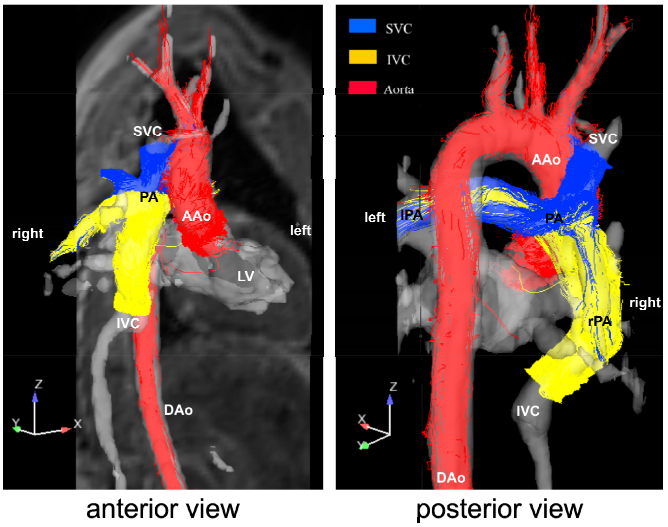


Fig. 1: **D-TGA**: 3D particle traces originating from emitter planes in the ascending aorta (AAo) and in the pulmonary artery (PA) represent blood flow velocities. Flow acceleration over 1.5m/s in left (lPA) and right (rPA) pulmonary artery can clearly be appreciated (white arrows) whereas the flow pattern in the aorta is normal.

Fig. 2, below: **TCPC in single ventricle**: 3D flow connectivity mapping of blood flow contributing to the filling of the aorta (AAo, red traces) and pulmonary system (yellow and blue traces) in a single ventricle patient after TCPC.



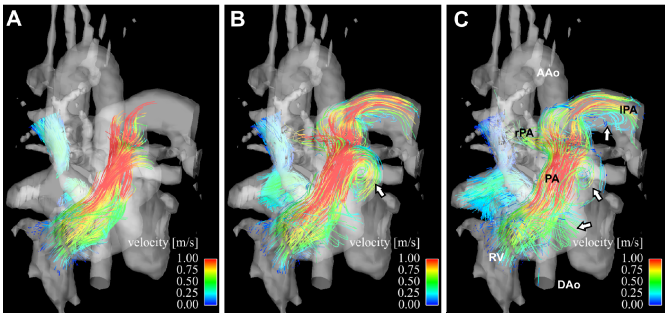


Fig. 3: **Tetralogy of Fallot**: 3D particle trace visualization for 3 systolic time-frames illustrates vortex formation (arrows) in the right ventricular outflow tract (A, B), the main pulmonary artery (PA) and the left PA (lPA) due to pronounced retrograde flow resulting in ectasia of the outflow tract (B, C).

Pat.	Cardiac defect	MR-finding
1-2	D-TGA (n=2)	Flow acceleration in both pulmonary arteries, normal aorta & slight retrograde flow (n=1)
3-4	Hypoplastic left heart, Norwood 2 (n=2)	Symmetric filling of rPA and lPA from SVC (n=1), systolic vortex and dilatation in aorta (n=2)
5	Hypoplastic left heart, Norwood 2, ISTA	Flow in SVC and IVC not evaluable, flow acceleration in coarctatio
6	Situs inversus, Glenn operation	Symmetric filling of rPA and lPA from SVC, filling hampered by antegrade flow, vortex, pulsatile flow in IVC
7	VSD, pulmonary stenosis	Severe retrograde flow in PA, vortex in RV, PA and rPA, ectasia of RVOT, flow acceleration in stenosis
8	Single ventricle, modified Fontan-Björk	Symmetric filling of rPA and lPA from SVC, slow flow in IVC to lPA, vortex in SVC to PA
9	Single ventricle, TCPC	Symmetric filling of rPA and lPA from SVC, symmetric filling of PA from IVC, vortex in aortic arch
10	Atresia of mitral valve, TCPC	Predominant flow from SVC to lPA, predominant flow from IVC to rPA
11-15	Repair of tetralogy of Fallot (n=5)	Moderate (n=1) to severe retrograde flow in PA (n=4), vortex in PA (n=3) and RV (n=2), RVOT ectasia (n=1)

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